

RESTRICTED CIRCULATION

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AGRICULTURAL MEMORANDUM NO 943

Newcastle disease - Part 2 - The 1969 Kent Outbreak

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INTRODUCTION

Gloster (1982) concluded that studies of past outbreaks of Newcastle disease, (ND) or fowl pest as it is more usually known, would be needed to confirm that airborne transmission of disease can occur in the field.

This memorandum examines one of these outbreaks - Kent 1969. This outbreak was chosen because:-

- (1) Disease information was available.
- (2) A velogenic virus strain was involved. This strain, latter called Essex '70 was known to cause widespread disease and high mortality.
- (3) Investigations made at the time of the outbreak found no link between any of the farms involved, Gilmore, (1971).
- (4) Infection via airborne virus had been already suggested for this outbreak, Gilmore (1971).

ASSUMPTIONS USED IN THIS STUDY

After discussions with W H Allan, Central Veterinary Laboratory, Ministry of Agriculture, Fisheries and Food (MAFF), Weybridge the following assumptions have been made:-

- | | |
|----------|--|
| ND virus | <ol style="list-style-type: none">(1) was excreted into the atmosphere by infected birds during this outbreak.(2) excretion was proportional to morbidity. ie the more severe the symptoms the greater the virus excretion. <u>It is interesting to note that even vaccinated birds excrete virus, but at very low levels. The level varies inversely with the birds immune titres⁷.</u> |
|----------|--|

- (3) excretion was proportional to the mass of the affected bird. ie. point-of-kill broilers excreted much more virus than day old chicks.
- (4) excretion started 2 days prior to the appearance of the first clinical symptoms.
- (5) remained airborne for a least an hour.
- (6) remained viable for at least an hour.
- (7) Infection occurred via the respiratory system.
- (8) Infection was most likely to occur 4-6 days prior to the appearance of the first clinical symptoms.

DISEASE SUMMARY

The great majority of disease information used in this paper has been obtained from J D Gilmore, MAFF, Maidstone.

During the 6 weeks, 10th February to 26th March 1969, a small epidemic of ND occurred in Kent. There were 8 outbreaks, more than had happened in Kent in a single epidemic since 1963. Disease was severe in unvaccinated birds, resulting in high mortality. The outbreaks occurred in the north-west corner of the county inside the Greater London boundary where there is a fair amount of urban development and poultry premises sparsely situated.

Figure 1 shows the relative distribution of the outbreaks and Table 1 the temporal distribution. Full details of each of the outbreaks are given below.

Outbreak 1

The first outbreak was in a commercial laying flock of 35,000 birds in controlled-environment deep litter houses. 25,000 adult birds had full vaccination cover and the remaining 10,000 younger birds only partial cover. The adult birds were affected primarily and they showed mild symptoms, minimal egg loss and a mortality of $1\frac{1}{2}\%$. Disease was about three weeks old when notified to the Ministry on 6-2-69.

The disease origin for this flock was most probably via infected crates which had been brought from the owners poultry processing plant in Lincolnshire. This

plant was in an area with confirmed disease outbreaks.

Outbreak 2

An unvaccinated broiler farm of 27,000 seven week old birds. First symptoms appeared on 6-2-69 and the disease was confirmed on 13-2-69. Losses were severe with all but 55 birds dying or were culled over a 23 day period. The carcasses were stacked outside of the buildings and left for a period of several weeks prior to disposal by the local authorities.

Outbreak 3

This outbreak occurred in a free range unvaccinated unit with a total stock of 300 one to two year old birds. First symptoms were seen on 14-2-69 and the disease was confirmed on either the 14-2-69 or 16-2-69. Six birds were found dead on the 14-2-69 and a total of 83% of the stock died over 25 days. The birds were kept in two groups and both groups were equally affected.

Outbreak 4

A 46,000 strong unvaccinated broiler unit. First symptoms were apparent on 16-2-69 and disease was reported to MAFF on 19-2-69. All of a house of 22,000 died within 2 weeks. In another group of 3,800 birds there was a mortality of 56%. There were 5 units on the farm. This farm was part of the same broiler unit as outbreak 2 but no form of contact between the two premises was found despite careful inquiries. Laboratory tests on birds from this farm showed that the virus was a velogenic form (ICPI = 1.71 and IVPI = 2.29. A definition of these units is given in Appendix 1).

Outbreak 5

A farm consisting of 4,500 fully vaccinated birds. First symptoms were noticed on 17-2-69 and the outbreak was reported on 21-2-69. Disease was very mild and there was no mortality.

Outbreak 6

This outbreak occurred amongst 2100 partly vaccinated layers. First symptoms were seen on 24-2-69 and were reported on 28-2-69. 1000 birds had full vaccination cover. Another group of 600 birds had not been vaccinated and here disease was severe (83% mortality). In a third group which were not fully protected there was a mortality of 40%.

Outbreak 7

1,700 fully vaccinated layers were on this farm. First symptoms were seen on 4-3-69 and it was reported on 6-3-69. Disease was very mild with only 1% mortality. Laboratory tests once again confirmed that the virus was a velogenic strain (ICPI = 1.67 and IVPI = 2.28).

Outbreak 8

The final outbreak in the series occurred on a farm of 1,000 fully vaccinated layers. First symptoms were seen on 9-3-69 but disease was not finally confirmed until 26-3-69. Mild symptoms were observed, including a fall of egg production on 9-3-69 but with no mortality.

ANALYSIS OF EPIDEMIC

START of the Epidemic

It has been pointed out previously that disease appears to have been introduced on to the first farm OUTBREAK NO 1/ON 1, via infected poultry crates, from the owner's poultry processing plant in Lincolnshire. Disease appears to have smouldered for several weeks at very low levels, (due to the protection given by vaccination), before being reported to MAFF. Symptoms at ON 1 were very mild and mortality only 1½%. Assuming that virus excretion into the atmosphere was proportional to the severity of the disease symptoms, it would appear that only small quantities of virus would have been excreted. If airborne virus was the cause for disease spread in this epidemic it would appear unlikely that ON 1 was the most likely source.

ON 2, only 75 m from ON 1 was situated at the road end of the same lane. Whilst examination of the wind data, taken 25 km away at Gatwick Airport, showed that there were no high risk wind conditions which might have carried virus from ON 1 to ON 2 for many days prior to infection, the possibility cannot be excluded entirely. If virus was introduced to ON 2 via the airborne route the most likely time would have been early morning on 29th January (8 days prior to first symptoms). On this occasion there was a period of 9 hours when the wind was reported as calm at Gatwick; it is quite possible that at ON 1 and ON 2 the winds could have been light in

strength and variable in direction. It is therefore possible that virus excreted from ON 1 was blown towards ON 2. However it is equally possible, that movement of birds, crates etc along the lane were responsible for introduction of disease to ON 2.

INTRODUCTION OF ND VIRUS TO ON 3 - ON 8

Once birds become infected at ON 2 and disease became established large quantities of virus would have been excreted into the atmosphere; a large number ($\rightarrow$ 22,000) of 7 week old birds with no vaccinal protection and almost total mortality were involved. Taking virus excretion as a function of morbidity it would have been at a low level on 4-2-69 (2 days prior to first clinical symptoms) and increased to a peak around the middle of the month before decreasing to zero by the end of the month.

Further appreciable quantities of virus would have been released into the atmosphere from ON 3, ON 4 and ON 6. In all cases there was high mortality.

Table 2 gives the dates for possible introduction of infection to ON 3-8 and the potential virus sources available during the 4-6 days susceptible period. The meteorological conditions during each of these periods is described in relation to transport of airborne virus from each of the potential sources to the subsequently affected farms.

AFFECTED FARM: ON 3

POTENTIAL SOURCE OF VIRUS: ON 2

PERIOD: 8-10th February

Overnight on 8/9th February there was a period of 15 hours when virus would have blown towards ON 3 from ON 2. The winds were light in strength (3 m sec^{-1}) and the lowest layer of the atmosphere stable; both of these conditions favour limited virus dispersion and hence high risk of subsequent infections. The relative humidity (RH) ranged from 67% at the start of the period to 91% in the early hours of 9th February.

AFFECTED FARM: ON 4

POTENTIAL SOURCE OF VIRUS: ON 2

PERIOD: 10-12th February

There were three periods when the wind was generally in the required direction for bringing virus to ON 4 from ON 2. From the evening of 9th February to 06.00 on 10th February, the windspeed ranged from 2-3.5 m sec⁻¹, the air was stable and the RH in the mid eighties (%). There was another period, of a few hours, later on the 10th when the windspeed was 2 m sec⁻¹ with neutral stability and a RH of 72%. During the final period, from 15.00 on 11th February to 09.00 on 12th February, the windspeed ranged from 3.5-6 m sec⁻¹, there was neutral stability and RH ranged from 77-85%.

AFFECTED FARM: ON 5

POTENTIAL SOURCE OF VIRUS: ON 2

PERIOD: 11-13th February

There were no clear cut periods when virus would have blown towards ON 5 from ON 2. However, virus could have drifted towards ON 2 during a period of reported calm (Gatwick) which lasted for 9 hours.

AFFECTED FARM: ON 6

POTENTIAL SOURCE OF VIRUS: ON 2

PERIOD: 18-20th February

Overnight on 17/18th February the conditions were suitable for virus to be blown towards ON 6 from ON 2. Wind speeds during this period were very low and were associated with misty conditions.

AFFECTED FARM: ON 6

POTENTIAL SOURCE OF VIRUS: ON 3

PERIOD: 18-20th February

After a period of calm on 18th there followed 39 consecutive hours of winds from ON 3 towards ON 6. Windspeeds ranged from 4-11 m sec⁻¹ and RH from 69-98%. Throughout most of the period there was total cloud cover and at times moderate snow.

AFFECTED FARM: ON 6

POTENTIAL SOURCE OF VIRUS: ON 4

PERIOD: 18-20th February

There were no periods between 18-20th February when the winds were favourable for transporting virus to ON 6 from ON 4.

AFFECTED FARM: ON 7

POTENTIAL SOURCE OF VIRUS: ON 6

PERIOD: 26-28th February

There were no occasions when the winds were favourable for transporting virus to ON 7 from ON 6.

AFFECTED FARM: ON 8

POTENTIAL SOURCE OF VIRUS: ON 6

PERIOD: 3-5th March

There were no occasions when the winds were favourable for transporting virus to ON 8 from ON 6.

Multiple disease cycles

From the previous analysis it can be deduced that by using a single 4-6 day infection period ON 3, 4 and 6 could be explained by the introduction of virus via the airborne route and that ON 5 was possible but less likely. The source of infection at ON 7 and 8 could not be explained.

It is noticeable that ON 5, 7 and 8 were the only forms which had full vaccinal cover. Disease on these premises was very mild and it is conceivable that it may have been present for some time prior to detection. If this were the case more than one disease cycle would have been involved.

Examination of the meteorological observations 10-11 days prior to disease being noticed on ON 5, ie two cycles, shows that there was a period of up to 9 hours when conditions favoured airborne transmission of virus from ON 2.

Favourable wind directions existed, from ON 3, 12-14 and 17 days prior to symptoms being detected on ON 7. Whilst a precise disease pattern is hard to determine for ON 3 (a free range unit) it would seem feasible for virus to be excreted from this farm to ON 7 over 0.5 km.

13-14 days prior to detection of symptoms at ON 8 the meteorological conditions over a 6 hour period favoured transport of virus from ON 4.

Summary of 4-6 day Infection Periods

Table 3 summarises the findings of this section. The possibility of airborne virus being the cause of introduction of disease onto each farm is rated; low, medium or high.

ESTIMATED VIRUS DOSAGE AT ON 3-8 FROM VIRUS EXCRETED AT ON 2

A modified version of the program written by Blackall and Gloster (1981) to calculate downwind dosages of foot and mouth virus was used to calculate the dosage of ND virus excretion from ON 2. The modification consisted of changing the sampling rate from that of an Ox to a chicken (700 gm chicken = 500 mL/MIN^{-1} Kohn, 1955).

Meteorological data combined with a simulated virus excretion pattern were used as input to the model. It was assumed that:-

- (1) two major disease cycles occurred, (2) affected birds excrete $3 \times 10^5 \text{ ELD}_{50} \text{ day}^{-1}$ (Allan, Private Communication)., and (3) the number of affected birds as a function of time was:

February											
DATE	4	5	6	7	8	9	10	11	12	13	14
No. of affected Birds.	70	80	90	100	120	300	800	900	1,100	1,200	1,400

Results

Figure 2 shows a map of inhaled dosages with contours of 0.01 ELD_{50} (from the computer output) for each day during the period and Table 4 gives a very approximate daily inhaled dosage for each of the outbreaks. The analysis has not included the effect of virus excreted when the winds were reported as calm. As a result of this inhaled dosages may be a slight underestimate.

The results suggest that very low dosages of particles are involved. Taking into account the accuracy of reported wind measurements and their frequency, birds on all farms (except ON 8) would have inhaled a minimum dosage of $\approx 0.01 \text{ ELD}_{50}$ on at least one occasion.

Whilst ON 4 is separated from the centre of the epidemic by 8 km it would appear very consistent with the other outbreaks in respect of inhaled dosages of virus. The relationship between susceptible flock size and the chance of infection has not yet been determined. However it would appear reasonable that the larger the unit the greater the chance of infection. ON 4 with 46,000 birds was the largest unit in the area. Disease spread at even greater ranges than 8 km could have been limited due to the sparsity of susceptible birds.

One other point which emerges from these calculations of inhaled dosages is that birds at ON 6 would have received a considerable dosage on 13 and 14th February: this would be consistent with infection occurring two disease cycles prior to detection. Combining this evidence with that from the previous analysis it would appear that infection could have occurred one or two cycles prior to detection of the first symptoms.

CONCLUSIONS

1. The significant proportion of the disease spread in this outbreak is consistent with an airborne hypothesis.
2. Airborne infection of non-vaccinated birds is associated with one 4-6 day infection cycle, prior to the observation of first symptoms.
3. Airborne infection on vaccinated birds is associated with two 4-6 day infection cycles, prior to the observation of first symptoms.
4. Airborne infection may have occurred up to 8 km from a virus source.
5. Very low downwind dosages were involved.

Acknowledgements

The author wishes to thank J D Gilmore and W H Allan for their invaluable help throughout the project.

References.

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Any comments or suggestions on this paper should be referred to the author.

Meteorological Office, London Road Bracknell, March 1982.

Berkshire RG12 2SZ

Appendix 1 - Definition of IVPI and ICPI

IVPI: The intravenous pathological index.

The index gives a simple estimate of the virulence of the sample under test. 6 week old chickens are exposed to virus and the number that die over a period are noted. Virus strains are identified on a scale 0 to 3; the more virulent the virus the greater the value. A value of 3 is obtained if all of the chickens die within 24 hours of exposure to virus and 0 if none of the chickens die within 15 days.

Lentogenic virus - Test not used to identify this virus type.

Mesogenic virus = 0 -0.5

Velogenic virus = > 0.6

Examples: Herts '33 approaches 3

Essex '70 2-2.6

ICPI: The intracerebral pathological index.

The index once again gives an estimate of the virulence of the virus under test. Day old chicks are exposed to the virus and the number that die over over a period are noted. Virus strains are identified on scale from 0 to 2; the more virulent the virus the greater the value.

Lentogenic virus = 0 -0.5

Mesogenic virus = 0.6-1.6

Velogenic virus = > 1.7

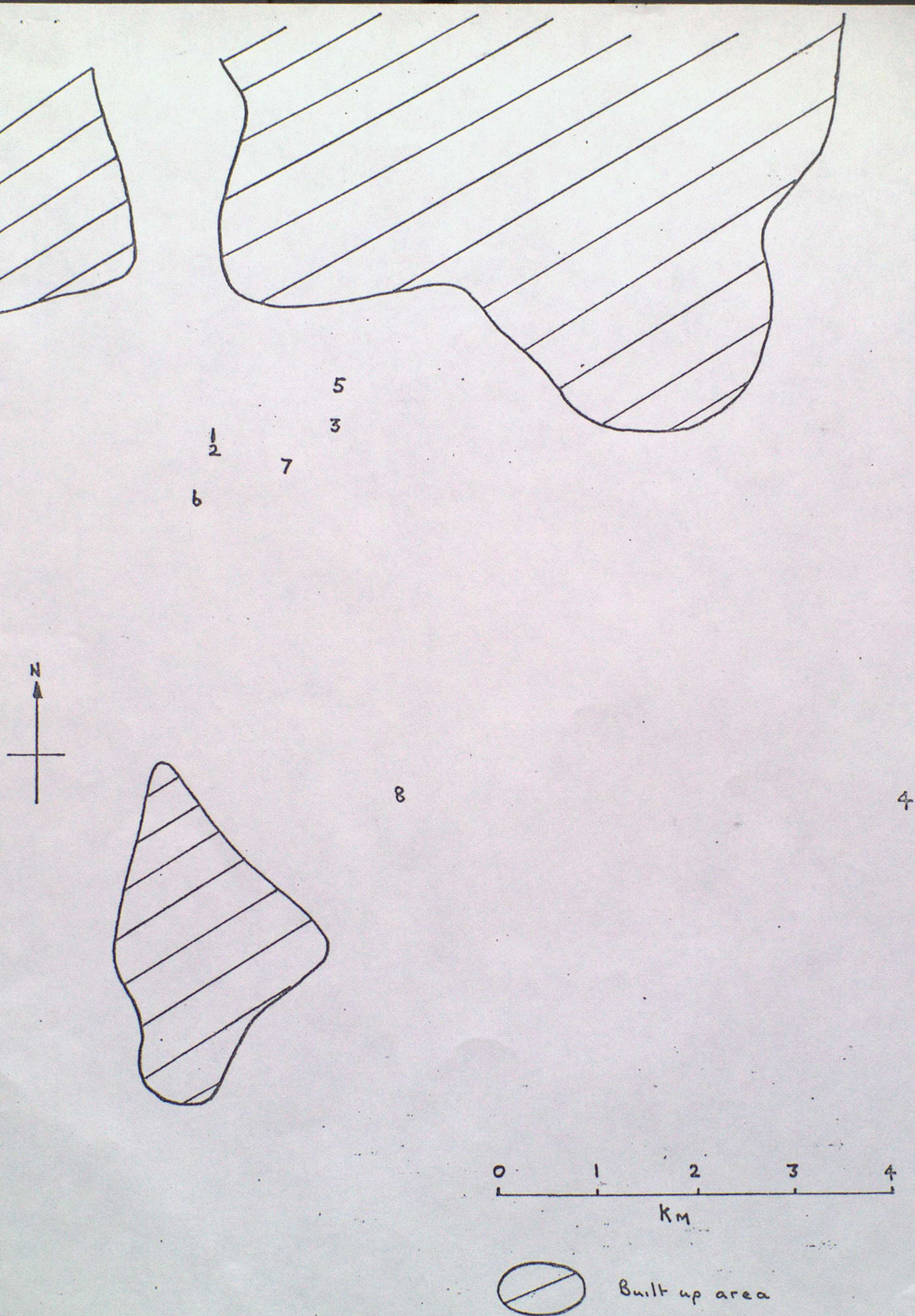
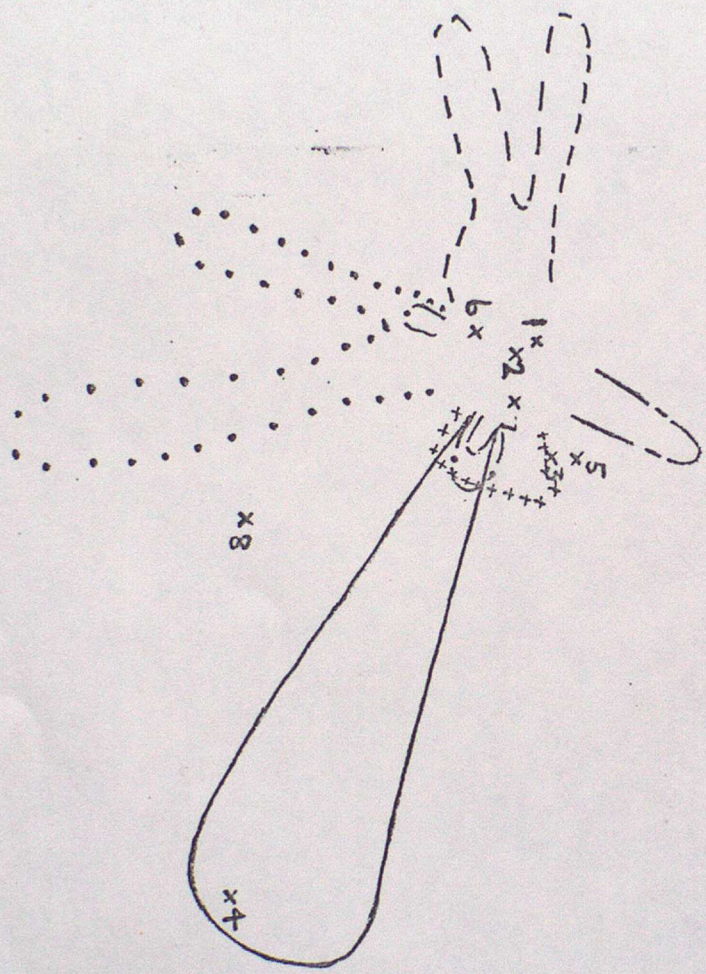
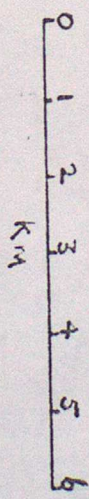
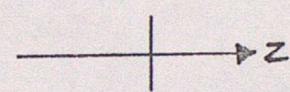


Figure 1. Relative positions of the Outbreaks



+++	9th February
---	10th "
---	11th "
---	12th "
...	13th "
---	14th "

Figure 2. Computer output. Contours of 0.01 ELD₅₀ throughout the period 9-14th February.
Source of virus Outbreak Number 2.

March

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$$R_{26/3}$$

R = Reported or
Confirmed

Table 1.
Temporal distribution of Outbreaks

Table 2. Possible dates for introduction of infection to Outbreak
No. 3-8 and the potential virus sources

Outbreak No	4-6 days Prior to first symptoms	Potential virus source (outbreak No.)
3	8-10 Feb	2
4	10-12 Feb	2
5	11-13 Feb	2
6	18-20 Feb	2, 3, 4
7	26-28 Feb	6
8	3-5 March	6

Table 3. The possibility of airborne infection at Outbreak No 3-8.

OUTBREAK NO.	VIRUS SOURCE (OUTBREAK NO.)			
	2	3	4	6
3 C_1 C_2	H			
4 C_1 C_2	H			
5 C_1 C_2	L M			
6 C_1 C_2	H	H		
7 C_1 C_2		M		
8 C_1 C_2			L	

C_1 One disease cycle

C_2 Two disease cycles

Possibility of Airborne infection

L = Low

M = Medium

H = High

Table 4. Estimated inhaled daily dosages ($ELD_{50} \times 10^{-2}$) of virus particles at Outbreak No. 3-8 from a virus source at Outbreak No. 2.

DATE: FEBRUARY

OUTBREAK	4	5	6	7	8	9	10	11	12	13	14
3	.02	.02	.01	.05	.1	1.2		.1			
4	.05				.01	.1	1	.6	.1		
5	.02		.02	.05		.01		.04			
6		.03					.08		1	8.0	3
7	.06	.05	.01	.1	.07	1.5	2	.8	.8		
8	.01	.02			.01		.1		.1	1.	

NOTE: where no value is given dosages were either too small to measure or zero.